Diagnostic and Interventional Imaging (2015) **96**, 37—43

ORIGINAL ARTICLE / *Musculoskeletal imaging*

An MRI evaluation of changes in piriformis muscle morphology induced by botulinum toxin injections in the treatment of piriformis syndrome

M. Al-Al-Shaikh [a], F. Michel [b,c], B. Parratte [d,e], B. Kastler [a,c], C. Vidal [f], S. Aubry [a,*,c]

[a] *Department of Osteoarticular Imaging, Besançon Regional University Hospital, hôpital Jean-Minjoz, 3, boulevard Alexandre-Fleming, 25030 Besançon cedex, France*
[b] *Department for Neuromuscular Disease and Exploration, Besançon Regional University Hospital, 3, boulevard Alexandre-Fleming, 25030, Besançon cedex, France*
[c] *Laboratory I4S, EA4268 IFR133, University of Franche-Comté, 25000 Besançon, France*
[d] *Department of Physical Medicine and Rehabilitation, Besançon Regional University Hospital, 3, boulevard Alexandre-Fleming, 25030 Besançon cedex, France*
[e] *Anatomy Laboratory, University of Franche-Comté, 25000 Besançon, France*
[f] *Clinical Investigation Centre, Besançon Regional University Hospital, 3, boulevard Alexandre-Fleming, 25030 Besançon cedex, France*

KEYWORDS
Piriformis syndrome;
MRI;
Botulinum toxin;
Piriformis muscle;
Obturator internus muscle

Abstract
Purpose: Botulinum toxin (BT) injection is a new treatment for piriformis syndrome (PS). The main purpose of our study was to use MRI to evaluate changes in piriformis muscle morphology after treatment with BT injections.
Patients and methods: Twenty patients presenting with PS who had undergone an MRI were included retrospectively: 12 patients treated with BT injections and eight untreated patients. The following parameters were assessed and compared to a normal contralateral muscle: maximum thickness, volume, and Goutallier's classification grade of fatty infiltration of the piriformis and internal obturator muscles. Pain was assessed through a visual analogue scale (VAS).
Results: The untreated patients had no significant difference in the volume ($P = 1.0$) or thickness of the piriformis muscle ($P = 0.61$). The treated patients showed a significant reduction in the thickness (−4.2 mm; $P < 0.001$) and volume (−74.4 mm^3; $P < 0.001$) and an increase in the fatty infiltration ($P < 0.001$) of the piriformis muscle treated by BT injection. Muscular atrophy

* Corresponding author.
E-mail address: saubry@chu-besancon.fr (S. Aubry).

2211-5684/$ — see front matter
http://dx.doi.org/10.1016/j.diii.2014.02.015

was correlated with the number of BT injections and with the time until an MRI was performed. There was also significant pain relief after BT treatment.
Conclusion: BT leads to atrophy and fatty degeneration of the piriformis muscle that can be quantified by MRI and these factors explain why BT injections are effective in the treatment of PS.

Sometimes buttock pain or pain beginning in the buttocks and radiating to the lower limb can originate in the piriformis muscle. This may be due to a problem in the muscle itself or to a permanent or intermittent compression of the trunk of the sciatic nerve (or ischiatic nerve) as it passes the piriformis muscle [1].

In terms of anatomy, the piriformis muscles have a triangular muscular body, with the base of each muscle inserted into each side of the ventral surface of the sacrum around the 2nd and 3rd sacral foramina. The piriformis muscle exits the pelvic cavity by sliding through the greater sciatic notch of the os coxae, above the sacrospinous ligament. It then crosses the gluteal region diagonally heading downwards to terminate at the superior border of the greater trochanter of the femur (Fig. 1). In the gluteal region, it is situated under the gluteus maximus muscle and above the termination of the obturator internus muscle and the gemellus muscles. This means that the piriformis muscle forms the border between two zones that muscles and ligaments pass through, known as the suprapiriform foramen and infrapiriform foramen. The superior gluteal vessels and nerves pass through the suprapiriform foramen. The sciatic nerve courses through the infrapiriform foramen, together with the inferior gluteal and pudendal nerves. There are anatomical variations in the course of the sciatic nerve through the infrapiriform foramen. These consist in particular of the muscle being penetrated by the tibial or common fibular nerves that form the sciatic nerve (11.7%). The tibial nerve can also pass above the piriformis muscle, through the suprapiriform foramen, while the common fibular nerve courses through the infrapiriform foramen (3.3%) [2]. More rarely, the undivided sciatic nerve can penetrate the piriformis muscle (0.8%) [2,3].

Figure 1. Posterior view of the right gluteal region. The sciatic nerve (arrow) passes through the infrapiriform foramen, bordered superiorly by the piriformis muscle (white arrow), and inferiorly by the portion of the obturator internus muscle that is outside the pelvis (white arrowhead).

Piriformis syndrome (PS) is sciatica that begins in the buttocks, or buttock pain that is secondary to compression of the sciatic nerve as it passes between the piriformis and obturator internus muscles. It poses problems both in terms of treatment and diagnosis, given that there is an absence of clinical or other signs that are pathognomonic. Chronic forms can be treated with botulinum toxin (BT) injections, and this is usually done with sonography guiding in the piriformis muscle or under CT guidance into the obturator internus muscle. Currently, the effect that botulinum toxin injections have on the muscle is poorly understood, and to our knowledge there is only one team that has published work using MRI to assess the muscle changes triggered by BT [4,5]. The main objective of this study is therefore to assess the effect of BT injections on the piriformis muscle (thickness, fatty degeneration, volume). The secondary objectives are to study the impact of the number of injections and the time from starting treatment to an MRI being carried out on the morphological parameters of muscles treated with BT, and to assess the efficacy of the treatment in terms of pain relief.

Patients and methods

This was a single-centre case-control study that did not require endorsement from an ethics committee because it was retrospective.

Definition of the disorder and recruitment

All of the subjects consulted a hospital doctor specialising in physical, sports, and rehabilitation medicine. During the interview, patients sometimes described pain on climbing stairs or walking uphill. There was often increased pain on rest. Buttock pain was felt, radiating through the thigh along the course of the sciatic nerve, but not below the knee,

mimicking sciatica due to disc herniation at the S1 nerve root [1]. In order to prove that these pains had a local muscular origin, a number of diagnostic manoeuvres were used including Freiberg's manoeuvre, Pace's manoeuvre [6], and the heel-contralateral knee manoeuvre [3].

A diagnosis of PS was made based on the association of the clinical arguments and signs on electroneuromyography (ENMG). The purpose of ENMG is to detect whether there is L5 or S1 nerve root involvement and to exclude nerve trunk involvement. In general, neither acute nor chronic neurogenic signs are found in PS. By contrast a nerve conduction study using the flexion—adduction—internal rotation manoeuvre [7] looks at the affected side for a delay in the conduction of the H reflex compared to a reference point with a threshold estimated at 1.8 ms [8]. Laboratory studies can be requested in order to exclude an inflammatory syndrome. Imaging studies (standard radiography, CT, MRI) of the spine, hips, and pelvis can exclude the differential diagnoses of PS and look for an extrinsic compression of the sciatic nerve. Recruitment was carried out retrospectively among all patients who had undergone MRI as part of investigations for PS between 10/12/2009 and 28/06/2012, a total of 23 subjects.

Inclusion and exclusion criteria

Criteria for inclusion and exclusion from the ''treatment'' group: We included patients who presented PS and had received BT injections in the piriformis muscle under sonographic guidance (Fig. 2) or under CT guidance into the piriformis and obturator internus muscles (Fig. 3). The doses injected were 100 IU of BT (Botox®) in one muscle. Patients who had been treated with surgical release of the piriformis muscle were excluded.

Inclusion criteria for the ''control'' group: patients presenting PS who had received neither treatment with BT injection nor surgical treatment.

Variables studied

In the treated patients, the number of botulinum toxin injections and the time between starting treatment with BT and an MRI being performed were documented. Pain was assessed using a visual analogue scale (VAS), with buttock pain (VASFess) and sciatic pain (VASSciat) routinely rated on the day of the MRI scan.

All subjects had 1.5 or 3 T MRI examinations (MRI 1.5 T Area Siemens, Erlangen, Germany and MRI 3 T HDX Signa Excite General Electric Medical System, USA). The protocol was as follows: pelvic MRI with spin echo T1-weighted acquisition of axial cross-sections (thickness: 5 mm, TE: 20 ms and TR: 860 ms), proton density-weighted fat-saturated (PD FatSat) axial and coronal cross-sections (thickness: 5 mm, TE: 13—35 ms and TR: 2200—2800 ms). The MRI studies were carried out on consoles equipped with the software Osirix (V 3.9.1 64 bit), which allowed for an assessment of the piriformis and obturator internus muscles in terms of the following criteria:

- quantitative values:
  - measure of the maximum transverse thickness in mm of the piriformis (TPir) and obturator internus (TObt) muscles (Fig. 4),

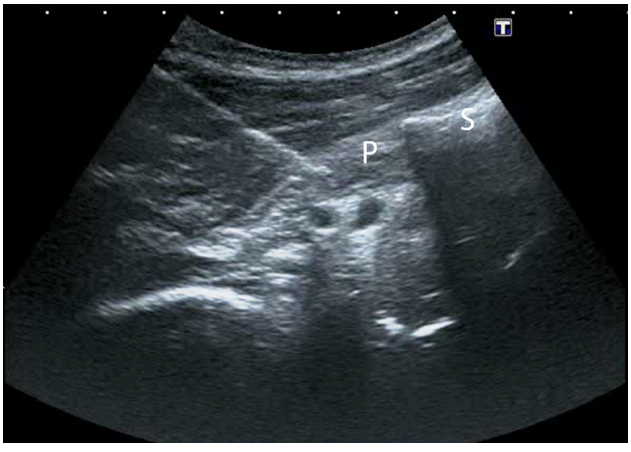

Figure 2. Sonographic cross-section taken during botulinum toxin (BT) injection into the piriformis muscle. P: piriformis muscle; S: sacrum.

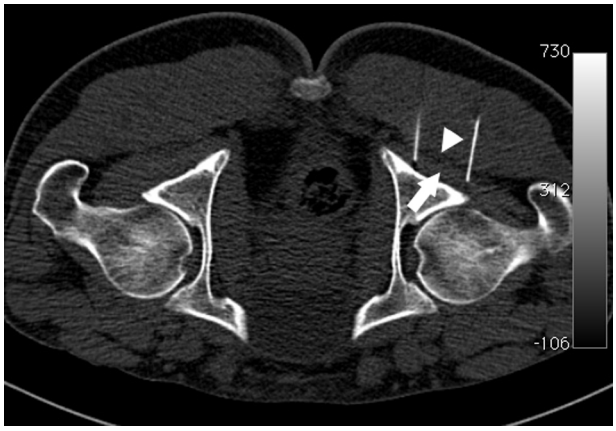

Figure 3. Cross-section from CT guidance during botulinum toxin (BT) injection into the portion of the obturator internus muscle that is outside the pelvis (arrow). Two needles are placed in the muscle, passing each side of the sciatic nerve (arrowhead). CT guidance was chosen over sonography because the obturator internus muscle is not particularly thick at this point and because of the proximity of the sciatic nerve.

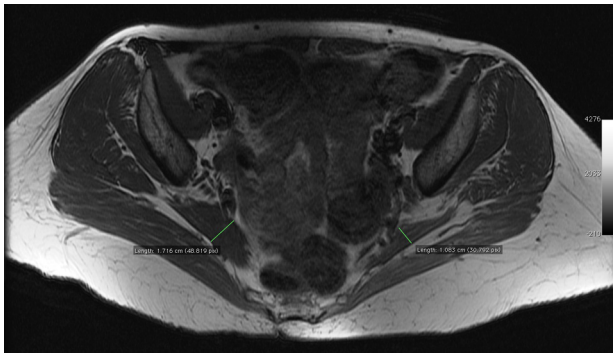

Figure 4. Axial T1-weighted cross-section. Thickness of the piriformis muscles measured in a 59-year old female treated with BT injection to the left piriformis muscle. Amyotrophy of the left piriformis (10.8-mm thick) compared to the right side (17.1-mm thick).

- calculation of the volume of the piriformis (VPir) and obturator internus (VObt) muscles and of the portion of the obturator internus outside the pelvis (VObtExo): segmentation was performed by manually delineating the contours of each anatomical structure of muscle sequentially cross-section by cross-section using the tool ''ROI of closed polygon'' then the volume was calculated in mm^3 using ''computed volume'' (Fig. 5);

- qualitative values:
  - assessment of the fatty infiltration of the piriformis (FIPir) and obturator internus (FIObt) muscles. Fatty infiltration was assessed using the five-grade Goutallier's classification [9]: grade 0 = no fat, grade 1 = streaks of fat, grade 2 = more muscle than fat, grade 3 = equal muscle and fat, grade 4 = more fat than muscle (Fig. 6),
  - looking for signs of acute denervation in the form of high-signal intensity within the muscles on FatSat PD-weighted images.

Statistical analysis

The statistical analyses were carried out using the software SAS for Windows (v9.3, SAS Institute Inc., Cary NC, USA). The mean (M) and standard deviation (SD) of the quantitative and qualitative values were calculated. For all tests, *P*-values below 0.05 were considered to be statistically significant. In the ''treatment'' and ''control'' populations the differences between the healthy and pathological side in terms of quantitative variables (TPir, TObt, VPir, VObt, VObtExo) were analysed using the paired Wilcoxon signed-rank test, and the differences in terms of qualitative variables (FIPir, FIObt) were analysed using the Kruskal-Wallis test. The correlation between the parameters of muscle morphology (thickness, volume, fatty infiltration) and the number of Botox® injections on the one hand and the time to an MRI being carried out on the other were studied by calculating Spearman's correlation coefficient (R). When there was a statistically significant correlation ($P < 0.05$), we used the following convention:

- $0 < |R| < 0.4$: weak correlation;
- $0.4 < |R| < 0.7$: moderate correlation;
- $0.7 < |R| < 1$: strong correlation.

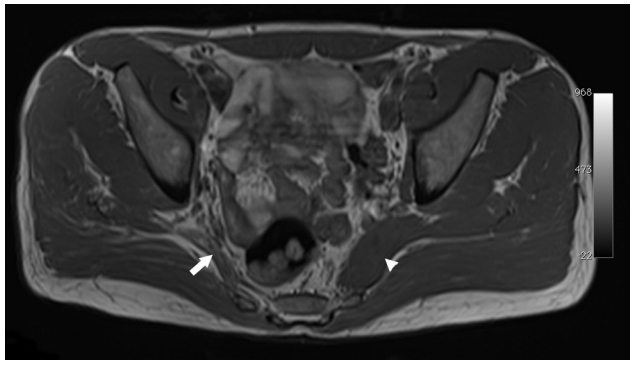

Figure 6. Grade 3 Goutallier's classification of fatty infiltration of the right piriformis muscle in a 42-year old patient treated with BT in the right piriformis muscle.

The differences in buttock pain and sciatic pain before and after treatment in the ''treatment'' population were analysed using the Wilcoxon signed-rank test.

Results

Description of the included population

Among the population of 23 subjects, three patients who had received surgical treatment were excluded. The treated subjects ($n = 12$) had a mean age of 46.5 years (min 26 years—max 63 years) and there was a strong preponderance of females (sex ratio: 2/10). Ten subjects had received BT injections in the piriformis muscle only, and two had received BT injections in the piriformis and obturator internus muscles. The time between starting treatment with BT and an MRI being performed was a mean of 7.3 months (SD = 5.2). Patients received 1—4 injections of BT (M = 2.1; SD = 1.1). The control group was made up of eight subjects with a mean age of 48 years (min 24 years—max 77 years) and once again there was a clear preponderance of females (sex ratio: 2/6).

Results from the ''control'' population

Based on the signed-rank paired test, there was no statistically significant difference in the variables VObt ($P = 0.054$),

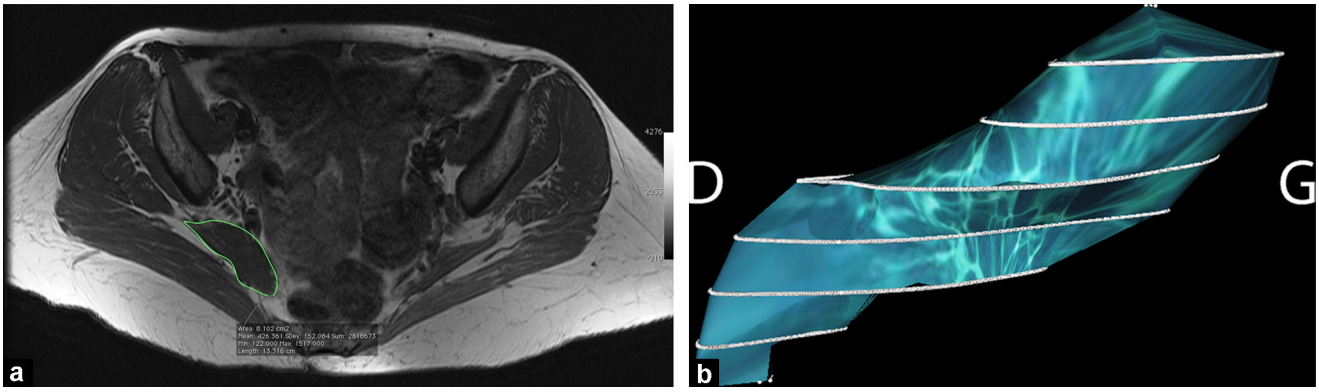

Figure 5. Manual segmentation of the right piriformis muscle on an axial T1-weighted cross-section (a) then 3D volume rendered view of the segmented piriformis muscle (anterior view) (b).

TObt ($P = 0.06$), VPir ($P = 1.0$), TPir ($P = 0.61$) or VObtExo ($P = 0.97$). This means that the mean of each of these variables is the same whether from the healthy side or pathological side in untreated patients.

Based on the Kruskal-Wallis test, we noted that there was no statistically significant difference in the grading of FIPir ($P = 0.73$) or FIObt ($P = 0.91$) between the pathological and healthy sides in untreated patients.

Results from the ''treatment'' population

Based on the signed-rank paired test, there was a statistically significant difference in VPir ($P < 0.001$) and TPir ($P < 0.001$) while the other values (VObt, VObtExo and TObt) did not show a significant difference. For example, VPir was 1.4 times greater on the healthy side than on the pathological side in treated patients (Table 1).

Based on the Kruskal-Wallis test, we noted that there was a statistically significant difference in the grading of FIPir ($P = 0.0002$) and FIObt ($P = 0.023$) between the pathological and healthy sides in treated patients.

Patients received a mean of 2.1 BT injections (min = 1, max = 4, SD = 1.11). We noted a strong correlation between TPir and the number of BT injections ($R = -0.77$, $P < 0.001$): as the number of BT injections increases, piriformis muscle thickness falls. We saw a moderate correlation between the number of BT injections and the fall in piriformis muscle volume ($R = -0.55$, $P = 0.003$). As the number of BT injections increases, the grading of the degree of fatty infiltration of the piriformis muscle increases ($R = 0.69$, $P < 0.001$).

The MRI scans were carried out a mean of 6.1 months after BT treatment was started. We noted a strong correlation between the length of time until an MRI was performed and a fall in TPir ($R = -0.69$, $P < 0.001$) while there was a significant but weak correlation between this timescale and a fall in VPir ($R = -0.39$, $P = 0.04$).

No treated patient presented any signs of acute denervation on the PD-FS sequence.

The subgroup of patients who received injections in the obturator internus muscle is too small ($n = 2$) to be considered individually for statistical analysis. We noted that on the treated side in these two patients, there was a mean TObt reduction of 2.4 mm, mean VObt fall of 30.7 mm^3, and mean VObtExo reduction of 32.3 mm^3, and both cases developed grade 1 fatty degeneration of the obturator internus.

Assessment of pain relief

There was a very significant difference ($P < 0.001$) on VAS scores between buttock pain before ($VAS = 7.50 \pm 1.61$) and after treatment ($VAS = 3.20 \pm 1.47$), and between sciatic pain before ($VAS = 7.04 \pm 1.05$) and after treatment ($VAS = 1.7 \pm 1.76$).

Discussion

Piriformis syndrome is defined as the compression of the sciatic nerve within the infrapiriform foramen [10]. The first description was made by Yeoman in 1928 but the credit for making this pure nerve compression syndrome into a true disease entity, and for naming it, goes to Robinson in 1947 [11]. Although it has been discussed for a long time, it is currently recognised as a genuine compression neuropathy due to clinical, anatomical, and electrical studies and because of the progress in modern imaging. Over the last few years there has been an increase in publications, especially in terms of information from MRI and regarding non-surgical treatment options [10]. This is one of the rare causes of sciatic pain that is not spinal in origin [12]. Although it has been established as a genuine compression neuropathy originating from the passage of the sciatic nerve through the infrapiriform foramen, there are various potential aetiologies for this compression. It may be due to inflammation, trauma, a tumour, a malformation [13,14], but most often it is muscular, and it is this latter aetiology that we addressed. There are a few studies that have shown that the obturator internus muscle may also potentially compress the sciatic nerve within the pelvis [2,3,15]. Since it forms the medial border of the infrapiriform foramen, a potential site of compression, entrapment between the piriformis and obturator internus muscles could be suspected [2,3].

MRI sometimes demonstrates hypertrophy of the piriformis muscle [16,17], an anatomical variation of the piriformis muscle or the course of the sciatic nerve [18–20], or a change in the sciatic nerve signal, and these changes affect their respective anatomical relationships with adjacent structures. One study showed that asymmetry of the piriformis muscles combined with a sciatic nerve signal abnormality had a specificity of 93% and sensitivity of 64% for predicting a good outcome to surgery on the piriformis muscle [21], but, in these surgically treated patients, 38.5% presented hypertrophy and 15% atrophy of the piriformis.

Table 1 Thickness (mm), volume (mm^3) and comparative test of the obturator internus and piriformis muscles on the healthy side versus the pathological side in treated patients. The *P*-values (<0.05) are in bold print.

Variables	*n*	Healthy side		Pathological side		Mean of the differences (pathological−healthy)	Paired signed-rank test *P*
		Mean	Standard deviation	Mean	Standard deviation		
VPir (mm^3)	12	254.08	60.90	179.66	54.76	**−74.42**	**<0.001**
TPir (mm)	12	16.21	1.61	11.99	2.15	**−4.22**	**<0.001**
VObt (mm^3)	12	333.14	80.47	329.98	69.73	−3.15	0.81
VObtExo (mm^3)	12	112.16	146.79	57.48	27.18	−54.68	0.22
TObt (mm)	12	10.97	1.65	9.80	1.52	−1.17	0.091

In addition, Russell et al. [22] used a population of 100 asymptomatic subjects (200 buttocks) to demonstrate that 90% of cases showed asymmetry in piriformis muscle thickness ranging from 3 to 8 mm. Moreover, there are true cases of PS in which the piriformis is of normal size [23]. In our ''control'' population, the absence of significant asymmetry in piriformis muscle thickness and volume argues in favour of this being a nerve compression that is perhaps not simply due to hypertrophy of the piriformis, which is not always present and has not been formally demonstrated, but one that is instead a functional compression of the sciatic nerve during muscle contraction. This absence of hypertrophy of the piriformis muscle could also be related to the very small sample size ($n = 8$). A role for the obturator internus muscle in PS has also been suggested [2]. Meknas et al. published a case series of six patients whose symptoms were relieved by surgical release of the obturator internus muscle [15], which led us to also inject this muscle in two patients, but the value of treating this site needs to be studied in larger populations.

The current chronological treatment plan for PS is based on firstly introducing analgesia (non-opioids and weak opioids) and muscle relaxants combined with structured physiotherapy, and if this management is ineffective or insufficient, there is recourse to intramuscular injections of botulinum toxin in the affected piriformis muscle [3,4,24—26]. Surgery to release the distal tendon is discussed in a few treatment-resistant cases.

Intramuscular botulinum toxin injections are used to reduce muscle overactivity, especially in the treatment of focal spasticity. Botulinum toxin blocks pre-synaptic conduction thus inhibiting cholinergic mediation and preventing the injected muscle from contracting, resulting in paresis of this muscle. The injection must be as close as possible to the motor end-plate. This leads to a beneficial effect on piriformis muscle contracture and it indirectly relieves sciatic pain. The clinical results from publications discussing use of BT seem to be very encouraging: Calvillo et al. [27] used 50 IU (12 good outcomes out of 14), Childers et al. [25] used 100 IU (excellent and very good outcomes in nine placebo-controlled cases), Yoon et al. [28] used 150 IU in 20 patients, and Michel et al. [3] used 50—100 IU in 250 patients whose results assessed by VAS were good and very good in 94 cases (77 %), moderate in eight cases (7.4 %) and poor in 19 cases (15.6 %). In our series, the efficacy of BT also translated into a very significant fall in VAS ratings post-injection ($P < 0.001$): we noted a reduction in buttock pain of around 50% after treatment, and a 75% fall in sciatic pain. Our results are therefore in agreement with data from the literature and together confirm the efficacy of botulinum toxin injection in PS [3,25,26]; these works, however, have focused purely on a clinical assessment of efficacy and only a single team to date has attempted to link this efficacy to changes in muscle morphology [4,5]. Our study shows that BT causes atrophy and fatty infiltration of the piriformis muscle. These results are in agreement with those from the nine patients assessed by MRI by Fanucci et al. [4,5], and they prove that:

- use of sonographic and electroneuromyographic landmarks meant that the BT injections in the piriformis were performed correctly;
- BT does indeed cause atrophy and fatty infiltration of the piriformis muscle secondary to muscular denervation.

The patients in our ''treatment'' population underwent MRI a mean of 6.1 months after they started BT treatment: this was for medium term follow-up, which explains why none of the patients presented any sign of acute denervation on the PD-weighted FatSat sequence. The phenomena of atrophy and fatty infiltration observed were relatively delayed and they correlated both with the number of BT injections and the timescale between the start of treatment and the MRI. Since the duration of action of BT is 3—5 months and the effect seems to be cumulative, in practice this prompts a repeat BT injection from the third month if painful symptoms recur or there is insufficient improvement: our patients received a mean of 2.1 BT injections. There is also a recommendation that the timescale of a minimum of three months between two injections should be respected in order to avoid sensitisation phenomena (allergy). Using this treatment plan, it seems that there is a place for follow-up MRI scans in patients whose symptoms are treatment-resistant in order to check what effect the BT has had on the muscle, and to assess muscle mass in view of potential additional injections.

The three surgical cases that were excluded from the series also presented atrophy (muscle volume fell by almost half: 228 mm^3 on the operated side as against 402.4 mm^3 on the healthy side) and fatty infiltration of the piriformis muscle (one case of grade 2 fatty infiltration, and two grade 3 cases), which can easily be understood because the muscle had been released. This treatment remains a last resort, on the condition that medical treatment has been tried and has failed, and that the diagnosis of PS is absolutely certain. The primary objective is to release the piriformis muscle by performing a tenotomy at its insertion on the greater trochanter.

The main forms of bias and the drawbacks to our study are: the possibility of selection bias that is inherent in the retrospective design of the study, and the sample size being too small to carry out a multivariate analysis. A further longitudinal analysis is needed in order to evaluate clinical efficacy in the longer term and to make a better assessment of the postulated link between changes in muscle morphology and clinical efficacy.

Conclusion

Pelvic MRI is currently the best examination for exploring the local anatomy, including the bone and muscle borders of the infrapiriform foramen, and for visualising the course of the sciatic nerve. Most importantly it allows for a quantitative assessment of changes in muscle morphology. Our study made a link between the efficacy of BT in the piriformis muscle for the treatment of PS and the atrophy and fatty infiltration that it triggers. This means that there seems to be a place for follow-up MRI scans in patients whose symptoms are treatment-resistant in order to check what effect BT has had on the treated muscle, and to assess the remaining muscle mass with a view to additional injections.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

References

[1] Erauso T, Pegorie A, Gaveau YM, Tardy D. Syndrome du muscle piriforme. Rev Prat 2010;60(7):900—4.

[2] Michel F, Decavel P, Toussirot E, Tatu L, Aleton E, Monnier G, et al. The piriformis muscle syndrome: an exploration of anatomical context, pathophysiological hypotheses and diagnostic criteria. Ann Phys Rehabil Med 2013;56(4):300—11.

[3] Michel F, Decavel P, Toussirot E, Tatu L, Aleton E, Monnier G, et al. Piriformis muscle syndrome: diagnostic criteria and treatment of a monocentric series of 250 patients. Ann Phys Rehabil Med 2013;56(5):371—83.

[4] Fanucci E, Masala S, Sodani G, Varrucciu V, Romagnoli A, Squillaci E, et al. CT-guided injection of botulinic toxin for percutaneous therapy of piriformis muscle syndrome with preliminary MRI results about denervative process. Eur Radiol 2001;11(12):2543—8.

[5] Fanucci E, Masala S, Squillaci E, Sodani G, Varrucciu V, Ursone A, et al. Pyriformis muscle syndrome: CT/MR findings in the percutaneous therapy with botulinic toxin. Radiol Med 2003;105(1—2):69—75.

[6] Pace JB, Nagle D. Piriform syndrome. West J Med 1976;124(6):435—9.

[7] Solheim LF, Siewers P, Paus B. The piriformis muscle syndrome. Sciatic nerve entrapment treated with section of the piriformis muscle. Acta Orthop Scand 1981;52(1):73—5.

[8] Fishman LM, Zybert PA. Electrophysiologic evidence of piriformis syndrome. Arch Phys Med Rehabil 1992;73(4):359—64.

[9] Goutallier D, Bernageau J, Patte D. L'évaluation par le scanner de la trophicité des muscles de la coiffe des rotateurs ayant une rupture tendineuse. Rev Chir Orthop Reparatrice Appar Mot 1989;75(Suppl. 1):126—7.

[10] Kouvalchouk JF, Bonnet JM, de Mondenard JP. Les syndrome du pyramidal. À propos de 4 cas traités chirurgicalement et revue de la littérature. Rev Chir Orthop Reparatrice Appar Mot 1996;82(7):647—57.

[11] Robinson DR. Pyriformis syndrome in relation to sciatic pain. Am J Surg 1947;73(3):355—8.

[12] Jroundi L, Quessar El A, Chakir N, Hassani El MR, Jiddane M. Le syndrome du muscle pyramidal : une cause rare de sciatique non discale. J Radiol 2003;84(6):715—7.

[13] Beauchesne RP, Schutzer SF. Myositis ossificans of the piriformis muscle: an unusual cause of piriformis syndrome. A case report. J Bone Joint Surg Am 1997;79(6):906—10.

[14] Picco AG, Parajua Pozo JL. The piriformis muscle syndrome due to pyomyositis. Med Clin (Barc) 1993;100(11):436—7.

[15] Meknas K, Christensen A, Johansen O. The internal obturator muscle may cause sciatic pain. Pain 2003;104(1—2):375—80.

[16] Rossi P, Cardinali P, Serrao M, Parisi L, Bianco F, De Bac S. Magnetic resonance imaging findings in piriformis syndrome: a case report. Arch Phys Med Rehabil 2001;82(4):519—21.

[17] Jankiewicz JJ, Hennrikus WL, Houkom JA. The appearance of the piriformis muscle syndrome in computed tomography and magnetic resonance imaging. A case report and review of the literature. Clin Orthop Relat Res 1991;262:205—9.

[18] Pećina M. Contribution to the etiological explanation of the piriformis syndrome. Acta Anat (Basel) 1979;105(2):181—7.

[19] Benzon HT, Katz JA, Benzon HA, Iqbal MS. Piriformis syndrome: anatomic considerations, a new injection technique, and a review of the literature. Anesthesiology 2003;98(6):1442—8.

[20] Petchprapa CN, Rosenberg ZS, Sconfienza LM, Cavalcanti CFA, Vieira RLR, Zember JS. MR imaging of entrapment neuropathies of the lower extremity. Part 1. The pelvis and hip. Radiographics 2010;30(4):983—1000.

[21] Filler AG, Haynes J, Jordan SE, Prager J, Villablanca JP, Farahani K, et al. Sciatica of nondisc origin and piriformis syndrome: diagnosis by magnetic resonance neurography and interventional magnetic resonance imaging with outcome study of resulting treatment. J Neurosurg Spine 2005;2(2):99—115.

[22] Russell JM, Kransdorf MJ, Bancroft LW, Peterson JJ, Berquist TH, Bridges MD. Magnetic resonance imaging of the sacral plexus and piriformis muscles. Skeletal Radiol 2008;37(8):709—13.

[23] Lee EY, Margherita AJ, Gierada DS, Narra VR. MRI of piriformis syndrome. AJR Am J Roentgenol 2004;183(1):63—4.

[24] Ade-Hall RA, Moore AP. Botulinum toxin type A in the treatment of lower limb spasticity in cerebral palsy. Cochrane Database Syst Rev 2000;2:CD001408.

[25] Childers MK, Wilson DJ, Gnatz SM, Conway RR, Sherman AK. Botulinum toxin type A use in piriformis muscle syndrome: a pilot study. Am J Phys Med Rehabil 2002;81(10):751—9.

[26] Fishman LM, Konnoth C, Rozner B. Botulinum neurotoxin type B and physical therapy in the treatment of piriformis syndrome: a dose-finding study. Am J Phys Med Rehabil 2004;83(1):42—50 [quiz 51—3].

[27] Calvillo O, Driver L, Truong A. Botulinum Toxin in the Treatment of Piriformis Syndrome. The Predictive Value of D-Tubocurare. Reg Anesth Pain Med 2001;26(2):102.

[28] Yoon SJ, Ho J, Kang HY, Lee SH, Kim KI, Shin WG, et al. Low-dose botulinum toxin type A for the treatment of refractory piriformis syndrome. Pharmacotherapy 2007;27(5):657—65.